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09/762,963	02/14/2001	Angel Lopez	A20-017	2022

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EXAMINER
PRASAD, SARADA C

ART UNIT	PAPER NUMBER
1646	

DATE MAILED: 08/13/2002 3

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application N .	Applicant(s)	
	09/762,963	LOPEZ ET AL.	
	Examiner	Art Unit	
	Sarada C Prasad	1646	

– The MAILING DATE f this communication appears on th cover sheet with the correspondence address –

Peri d f r Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *14 March 2001* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-31* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-31* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Pri rity under 35 U.S.C. §§ 119 and 120

13)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☒ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) *2* .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

Detailed Action

1. Priority to PCT/AU99/00659 (8/13/1999) of this application is acknowledged. Claims 1-31 are currently under consideration for examination.

Claim objections:

2. Claim 1 is objected to because of a typographical error in reciting 'monoclonal antibody of capable of inhibiting any one of ..'. This objection can be obviated by reciting 'monoclonal antibody capable of inhibiting any one of ..'.

Claim Rejections - 35 USC § 112-First paragraph

3. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

3a. Claims 21, 23-27, 31 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Deposit of Biological Material:

The specification sets forth for preparation of a monoclonal antibody to β_c of IL-3, IL-5, GM-CSF termed BION-1, designated ATCC HB-12525, with ATCC at 10181 University Boulevard, Manassas, Virginia, USA, The specification also asserts that these monoclonal antibodies bind to the common β_c receptor of these three cytokines.

Claims 21, 23-27, 31 recite the use of the BION-1 antibody produced by the hybridoma of ATCC Deposit No. HB-12525, with ATCC at 10181. Such monoclonal antibodies designated

BION-1, directed to the common receptor β_C chain of IL-3, GM-CSF, and IL-5 must be obtainable by a repeatable method set forth in the specification or otherwise be readily available to the public, and this deposit is essential to the claimed invention. Disclosure of deposits of hybridomas is acknowledged. However,

an affidavit or declaration by Applicants or someone associated with the patent owner who is in a position to make such assurances, or a statement by an attorney of record over his or her signature, stating

(a) that the ATCC deposit has been deposited under the Budapest treaty; and

(b) that it will be irrevocably and without restriction or condition be released to the public upon issuance of a patent that would satisfy the deposit requirement made herein. See 37 CFR 1.808, has not been submitted along with the application.

Further, the record must be clear that the deposit will be maintained in a public depository for a period of 30 years after the date of deposit, or 5 years after the last request for a sample, or for the enforceable life of a patent whichever is longer. See 37 CFR 1.806. If the deposit has not been made under the Budapest Treaty, then an affidavit or declaration by applicants or someone associated with the patent owner who is in a position to make such assurances, or a statement by an attorney of record over his or her signature must be made, stating that the deposit has been made at an acceptable depository and that the criteria set forth in 37 CFR 1.801-1.809, have not been met.

Amendment of the specification to disclose the date of deposit and the complete name and the address of the depository is required.

If the deposit was made after the effective filing date of the application for a patent in the United States, a verified statement is required from a person in a position to corroborate that the deposits described in the specification as filed are the same as that deposited in the depository. Corroboration may take the form of a showing of a claim of custody from applicant to the depository coupled with corroboration that the deposit is identical to the biological material described in the specification and in the applicants' possession at the time the application was filed. Applicants attention is directed to In re Lunduk, 773 F.2d. 1216, 227 USPQ 90 (CAFC 1985), and 37 CFR 1.801-1.809 for further information concerning deposit practice.

Scope of enablement

3b. Claims 1-12, 14-20, 22, and 24-30 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for an antibody which binds domain 4 or the F'-G' loop of β_c and inhibits binding of IL-5, IL-3, and GM-CSF, methods of producing the antibody by immunizing with domain 4, and methods of use to inhibit eosinophil activation and leukemic cell growth by IL-5, IL-3, and GM-CSF, does not reasonably provide enablement for any other agents, antibodies, or inhibitors of β_c or of any other cytokine receptors, or portions thereof, or use to treat asthma, or cancer, or inhibit any other condition. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make the invention commensurate in scope with these claims.

The factors considered when determining if the disclosure satisfies enablement requirement and whether any necessary experimentation is undue include, but are not limited to: 1) nature of the invention, 2) state of the prior art, 3) relative skill of those in the art, 4) level of predictability in the art, 5) existence of working examples, 6) breadth of claims, 7) amount of direction or guidance by the inventor, and 8) quantity of experimentation needed to make or use the invention. *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988).

The specification sets forth for methods of isolation of a monoclonal antibody (BION-1) raised against the membrane proximal domain (domain 4) of β_c, which is able to block the production, and activation of human eosinophils stimulated by IL-5, GM-CSF, or IL-3 and blocks the growth of leukemic cell lines. This monoclonal antibody was able to block the high affinity binding of all three cytokines to eosinophils by binding to residues in the predicted B'-C' and F'-G' loops of β_c and prevent receptor dimerization and β_c phosphorylation (page 3 of specification, summary of the invention).

The claims are broad in the recitation of --generation of antibodies to an analogous domain in an analogous common receptor, or part thereof, or to a receptor analogous to β_C, or to a full or partial portion of domain 4 when it is in a configuration where the F-G loop and/or the B-C loop is in its native shape, or to an equivalent domain in other cytokine receptors, or to an analogous receptor/common receptor, which is any one of the cytokine superfamily receptors from the group including LIFR, gp130, IL-2Rβ, IL-R/IL-13R, IL-2Rγ, IL-3Rα, EPOR, TPOR, and OBR.

Specification discloses that the term "analogous" includes a number and variety of receptors with broad scope including not only the β_C chain of the receptor for GM-CSF, IL-3, and IL-5 but also including other receptors which belong to the rapidly expanding cytokine receptor super family, some of which are characterized by the sharing of a communal receptor subunit by multiple ligands.....' (page 3, 3rd para, lines 4-end). In the context of the scope of such communal receptors, the differences between the instant β_C receptor, and any other common receptor makes it unpredictable that only structural/functional features of the Bc receptor would be directly extrapolated with any other system, which renders the guidance provided insufficient to determine what regions on other receptors would have properties similar to β_C receptor. Indeed, the instant specifcation acknowledges that it is not known 'which residues in gp130, LIFR, and IL-2R β and γ chains are important for ligand binding or indeed whether different ligands share or have unique sets of binding determinants on these communal receptor sub-units .....'(page 4 of specification, line 2-end of para).

Additionally, not any part of the instant β_C receptor would predictably result in the generation of antibodies with desired specificities/properties. Only immunization with 'the F-G

loop of the domain 4' would predictably result in an antibody with the expected ability to inhibit the binding of IL-3, IL-5, and GM-CSF to the common receptor β_C, and not any other parts of it or domains of it. There is no guidance regarding portions, or regions other than the F'-C' and B'-C' which would predictably generate such antibodies. Furthermore, WO/9728190 discloses that B'-C' loop of β_C plays some role in high affinity binding of GM-CSF, IL-3, and IL-5, however, targeting of B-C loop by itself does not result in inhibiting the binding of all of the three cytokines to β_C receptor (page 4, lines 21-26) which limitation is encompassed by the phrase 'portion of the recepor' in the instant claims, and the recitations of B'-C' loop in claims 5 and 12.

Claims 17 and 20 are extremely broad in recitation of several cell types as the effector cells that the instant monoclonal antibody would be able to act as an effector of . The question of lack of enablement in this instance is does one of skill have the knowledge that all of these cells possess the β_C receptor, or if they are responsive to any one of the cytokines such as IL-3, or IL-5 or GM-CSF that act via β_C receptor. Watanabe et al. (1992) disclose that because β_C receptor is an essential component for signal transduction for IL-3, IL-5 and GM-CSF, the expression of β_C receptor should be critical for responding to antibodies to common receptor. In the absence of such data or guidance as to how one would proceed to test if all of these cells exhibit the common receptor β_C or other common receptor, and why they would be expected to be responsive to the instant cytokines or antibodies. Teachings of Watanabe et al. also include that the factors affecting the expression of β_C receptor may have potential to regulate the development of hematopoietic cells (page 2219, column 2, last 3 lines). Therefore, the instant specification is non enabling for monoclonal antibodies that inhibit binding of cytokines to cell

lines recited in claim 20, or cell lines referenced as effector cell lines in claim 17. Additionally, Figure 14 of specification teaches that growth *in vitro* of chronic myelomonocytic cells (CMML) is inhibited by BION-1, and the specification is only enabling for CMML in addition to eosinophils as expressing β_C receptor, and not all of the other cells recited in or encompassed by claims 17 and 20.

Claims 18 and 19 are extremely broad in reciting 'an antibody or a fragment thereof is used for treatment of asthma and leads to inhibition of IL-5, IL-3, and GM-CSF mediated eosinophil survival'. The specification only discloses support for inhibition of eosinophil activation and no guidance is provided with respect to treatment of asthma. It is noted that specification contemplates on how such blocking of the binding of these three cytokines to their common β_C receptor will be therapeutically useful for the intervention in conditions .....including asthma...(specification, page 9, 3rd Para). However, a review of "Eosinophils, eosinophilic cytokines and antieosinophilic therapy in asthma" by Menzies-Gow et al. reports that while eosinophils are believed to be key effector cells in producing the .....characteristic of allergic asthma, while eosinophils have been logical therapeutic targets, treatments at achieving effective depletion of eosinophils did not result in clinical improvement in any of the parameters studied, and the role of eosinophils in allergic inflammation was questioned (entire abstract). Therefore, inhibition of eosinophil survival and activation mediated by IL-5, IL-3 and GM-CSF alone is not sufficient criterion for effective treatment of asthma. No additional guidance is provided in the specification.

Claims 24-26 are broad in recitation of 'a method of isolating an inhibitor capable of competitively inhibiting the binding of BION-1, or the binding of an agent capable of inhibiting

BION-1 binding to the β_C receptor.....'. The specification is enabled for screening for inhibitors capable of inhibiting the binding of BION-1 to the β_C receptor, but the specification has not identified, nor is enabled for any inhibitors or agents capable of inhibiting inhibitors of BION-1 binding to the β_C receptor', because claim 24 is also drawn to identifying inhibitors of inhibitors using the very same steps. The method is a screening method to identify inhibitors of BION-1 binding, practice of the steps will not result in identification of anything else. It would require undue experimentation for one of skill to identify such compounds.

Claims 24-30 are broadly drawn to 'cytokine inhibitors' that simultaneously block the binding of IL-3, IL-5, GM-CSF to β_C receptor. The claim language can be interpreted to include inhibitors other than antibodies. The specification is not enabling for any inhibitors other than antibodies to domain 4 of the β_C receptor that is specific to the three cytokines, namely IL-3, IL-5, and GM-CSF. The guidance provided in the specification for one of skill to envision inhibitors other than antibodies, which would block binding of β_C receptor, or a fragment thereof to IL-3, IL-5, GM-CSF, and thus inhibit leukemic cell proliferation is for possible mimetic peptides of β_C receptor, as in Figure 13. However, neither of these peptides were shown to be effective inhibitors of binding as the difference between the 'no-peptide control' and 'the inhibitor' was marginal. This would be an example of the level of unpredictability in the art that any peptides or nucleotides would not be able to serve as inhibitors of cytokine binding to common receptors. Additionally, WO 97/28190 points out how 'potential cytokine inhibitors' can not be characterized by a mere binding to β_C receptor alone, because it is not the entire β_C receptor that would determine the binding of each of the three cytokines. Each of the three cytokines, IL-3, IL-5, and GM-CSF, have distinct requirements for binding to the common

receptor, such as "Tyr ^{365}, His ^{367}, and Ile ^{368} are important for GM-CSF and IL-5 high affinity binding, but are only marjinally involved in IL-3 high affinity binding, implying that targetting these three amino acids with appropriate compounds may impair GM-CSF and IL-5 but not IL-3 mediated activity" (page 4, 3rd para, lines 22-end). Based on molecular modeling techniques, it has been postulated that F'-G' loop of β_C receptor is essential for the high affinity binding of all three cytokines GM-CSF, IL-5 and IL-3 (page 4, lines 31-34). WO 97/28190 also discloses that "it is believed that compounds that bind to Tyr ^{421} or inhibit the binding of ligand to Tyr ^{421} will behave as generic antagonists of IL-3, GM-CSF, and IL-5 and the compounds that bind to the F'-G' loop will sterically inhibit this binding" (paragraphs bridging pages 4-5).

Written Description

3c. Claim 27 is rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Instant specification discloses screening of peptides for inhibition of MoAb BION-1 binding to soluble β_C domain 4 adsorbed to solid phase (Figure 13, specification, page 13). Two peptides namely, B45.pep and YB12.pep derived from biopanning libraries with the E.coli derived soluble β_C domain 4 ($s\beta_C$ #4) have not shown any significant difference in inhibiting the binding in comparison to (no-peptide) control in Figure 13.

In light of this disclosure, there is no actual reduction to practice of the claimed invention, or a clear depiction of the claimed invention. Weighting all the factors in view of the level of knowledge and skill in the art, one skilled in the art would not recognize from the

disclosure that the applicant was in possession of such inhibitors of BION-1 binding determined by the disclosed methods of screening.

Applicants must convey with reasonable clarity, to those skilled in the art, as of the filing date sought, he or she was in possession of the invention. Therefore, mere contemplation of claimed inhibitors in the absence of enabling description does not allow one skilled in the art to envision the claimed invention. Conception of the claimed invention cannot be achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the potential methods for screening for the inhibitors. Therefore the Applicants have not provided sufficient evidence that they were in possession of the invention at the time of filing, as it is claimed, and thus written description requirement has not been satisfied for the claims as they are recited.

Applicant's attention is drawn to Guidelines for the examination of patent Applicants under 35 U.S.C. 112 first paragraph, "Written Description" requirement, federal register, Vol.66, No. pages 1099-111, Friday January, 2001.

Claim Rejections - 35 USC § 112-Second paragraph

4. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claim 1-31 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claim 1 is incomplete for omitting essential steps, such omission amounting to a gap between the steps. See MPEP § 2172.01. The omitted steps are: For example, claim 1, while

claiming 'a method of isolating a monoclonal antibody...', in fact, fails to really isolate, it rather ends in screening.

Claim 8 is vague and indefinite due to recitation of '...inhibits binding of all the said receptors to a common receptor' because it is not clear as to what is 'receptor that binds to another receptor'.

Claim 9 is indefinite due to recitation of 'including' because the list of cytokines that follow the term 'including' may cover more cytokines than what are recited. The metes and bounds of the claimed invention cannot be determined.

Claim 16 is indefinite due to recitation of '...inhibition leads to blocking of at least one function of all three cytokines' because it is not clear what is that one function, and how is it defined. The metes and bounds of the claimed invention cannot be determined.

Claim 17 is indefinite due to recitation of 'activity leads to inhibition of or stimulation of effector cell activation, or survival' because it is not clear what is the effector cell? The term 'effector cell' appears to be used in a manner contrary to accepted definition of a cell that mediates an immune response versus what appears to be any cell that mediates an adverse condition of any kind which is indefinite, because the metes and bounds are indeterminate.

Claim 24 is indefinite due to recitation of 'measuring the degree of binding' because it is not clear as to 'the degree of binding' is relative to what. This rejection can be obviated by reciting the control for binding.

Claims 7 and 9 are vague and indefinite due to recitation of acronyms 'LIFR, gp130, EPOR, TPOR, and OBR because acronyms are subject to change and they may be used in more

than one instance. Therefore acronyms should be written in complete form when first used and then referenced at subsequent use.

Claims 1, 2, 6, 7, 8, 9, 24, 25, 29, and 31 are indefinite due to recitation of analogous domain, analogous receptor, etc. equivalent domain, common receptor, fragment thereof, because the metes and bounds of these terms cannot be determined. For example, the criteria that control whether something is analogous or not cannot be determined.

5. ***Obviousness type Double patenting:***

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970);and, *In re Thorington,* 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

5. Claims 1-20, 22, 28-30 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-5 of U.S. Patent No. 6,200,567 (2001).

Although the conflicting claims are not identical, they are not patentably distinct from each other, because claims 1-5 of U.S. Patent No. 6,200,567 (with one common inventor) recites an antibody that binds to the common β_c chain of the receptor for GM-CSF, IL-3, and IL-5, said common β_c chain having the amino acid sequence set forth in SEQ ID No. 1, wherein the said antibody antagonizes the effects of GM-CSF, IL-3, or IL-5 inhibiting the binding of the cytokine

to the common β_c chain with ability to inhibit any one of IL-3, GM-CSF, and IL-5 binding to the common β_c receptor. U.S. Patent No. 6,200,567 also teaches that the F-G loop of domain 4 of the common β_c receptor, a portion of the common receptor is critical for the high affinity binding and stimulation of function of GM-CSF, IL-3 and IL-5. These teachings would render instant claims 1-20, 22, 28-30 obvious.

Claim Rejections - 35 USC § 102

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

6a. Claims 1-20, 22, 28-30 are rejected under 35 U.S.C. 102(b) as being anticipated by WO 97/28190 (7/8/1997).

WO 97/28190 teaches that F'-G' loop of β_c chain of the receptor for GM-CSF, IL-5, IL-3 is essential for the high affinity binding and signaling of all the three cytokines (summary of the invention, page 4, 4th para, lines 3-5), and compounds that bind to the F'-G' loop of the β_c chain of the receptor will stearically inhibit binding of these three cytokines to the common β_c chain of the receptor (page 5, lines 3-4). Teachings of WO 97/28190 also include a therapeutic agent capable of binding to the F'-G' loop of domain 4 of the common β_c chain of the receptor for GM-CSF, IL-5, IL-3, and there can be many classes of compounds that fit the limitation of such therapeutic agents (page 8, 2nd para). WO 97/28190 also teaches the agent may be an antibody, or a fragment thereof, and the method of making such a monolconal antibody will include immunizing an animal with a peptide habing the F'-G' loop (paragraphs bridign pages 8-9).

Example 2 particularly discloses methods of making the blocking antibodies directed to the F'-G' loop of the β_c chain of the receptor that would inhibit the high affinity binding to GM-CSF, IL-5, IL-3, and inhibit stimulation of eosinophil activation (page 17, last para). These teachings meet the limitations of instnat claims 1-20, 22, 28-30.

6b. A person shall be entitled to a patent unless –

(e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

The changes made to 35 U.S.C. 102(e) by the American Inventors Protection Act of 1999 (AIPA) do not apply to the examination of this application as the application being examined was not (1) filed on or after November 29, 2000, or (2) voluntarily published under 35 U.S.C. 122(b). Therefore, this application is examined under 35 U.S.C. 102(e) prior to the amendment by the AIPA (pre-AIPA 35 U.S.C. 102(e)).

6b. Claims 1-20, 22, 28-30 are rejected under 35 U.S.C. 102(e) as being anticipated by U.S. Patent No. 6,200,567 (Mar, 2001).

U.S. Patent No. 6,200,567 teaches that F'-G' loop of β_c chain for GM-CSF, IL-5, IL-3 is essential for the high affinity binding, and signaling of all three cytokines (summary of the invention, page 4, 4th para, lines 3-5), and compounds that bind to the F'-G' loop of β_c chain will sterically inhibit binding of these three cytokines to their common β_c receptor (page 5, lines 3-4). Teachings of U.S. Patent No. 6,200,567 also include a therapeutic agent capable of binding to the F-G' loop of domain 4 of the common β_c chain of the receptor for GM-CSF, IL-5, IL-3, and there can be many classes of compounds that fit the limitation of such therapeutic agents (page 8, 2nd para). U.S. Patent No. 6,200,567 also teaches the agent may be an antibody, or a fragment

thereof, and the method of making such a monoclonal antibody will include immunizing an animal with the a peptide having the F'-G' loop (paragraphs bridging pages 8-9). Example 2 particularly discloses methods of making the blocking antibodies directed to the F'-G' loop of the β_c chain of the receptor that would inhibit the high affinity binding to GM-CSF, IL-5, IL-3, and inhibit stimulation of eosinophil activation (page 17, last para). These teachings meet the limitations of instant claims 1-20, 22, 28-30.

The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

Watanabe et al. (1992) Blood, vol.80, pages 2215-2220..

Watanable et al. teach three different monoclonal antibodies (CRS1, CRS2, and CRS3) against the common β_c receptor of the hIL-3, hGM-CSF, and hIL-5 using NIH3T3 transfectants expressing the high-affinity GM-CSF receptors. All three MoAbs bound to NIH/$\alpha\beta_c$ but not to NIH3T3. Watanabe et al. also taught the differences between CRS1 and CRS 2 and 3 in that CRS1 recognized both NIH/β_c, and NIH/$\alpha\beta_c$, while CRS2 and CSR3 bind only NIH/$\alpha\beta_c$.(page 2218, columns 1-2, discussion). Watanabe's teachings also include that these monoclonal antibodies would be useful in studying the pathogenesis of myeloid leukemic cells, because the common receptor is expressed on myeloid progenitor cells, as well as the fact that myeloid leukemic cells respond to IL-3, IL-5 and GM-CSF (page 2219, columns 2-3). Watanabe et al. did not teach BION-1 antibodies directed to the F'-G'loop of the /β_c receptor .

Conclusion

8a. Claims 24-27, and 31 are objected to insofar as they depend on rejected claims 1-20, 22, 28-30. 8b. No claims are allowed.

Advisory Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sarada C Prasad whose telephone number is 703-305-1009. The examiner can normally be reached Monday - Friday from 8.00 AM to 4.30 PM (Eastern time).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Yvonne Eyler, can be reached on (703) 308-6564. The fax phone number for the organization where this application or proceeding is assigned is 703-308-0294.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-0196.

Sarada Prasad, Ph.D.
Examiner,
Art Unit 1646
June 24th , 2002.

Yvonne Eyler

YVONNE EYLER, PH.D
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600